

# A guide to the clinical management of attrition

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## Key points

Discusses aetiology of attrition.

Discusses signs and symptoms of attrition.

Discusses clinical management of attrition including adhesive and conventional techniques.

Attrition is an enigmatic condition often found in older individuals and often as a result of bruxism which can take place as a result of either day bruxism, night bruxism or both. Various studies and systemic reviews clearly shown that tooth wear is an age-related phenomena and the last Adult Dental Health Survey showed that 15% of participants showed moderate wear and 3% severe wear with 80% of patients over 50 years of age showing signs of wear. This review examines current theories around the aetiological factors contributing to attrition together with the clinical management of attrition focusing on minimal intervention where possible.

## Introduction

Attrition is formally defined as the loss of tooth substance caused by tooth-to-tooth contact so although it is predominantly seen occlusally, attrition can also occur interproximally as lateral movement of the teeth produces broader interproximal contacts over time<sup>1</sup>(Fig. 1).

Typically, this type of wear is seen as marked wear facets with complimentary wear facets being seen in the upper and lower jaws. In very general terms, patients often tend to brux in an anterior/posterior direction or in a lateral direction. If they tend to brux anterior/posteriorly marked matching wear facets are often seen on the anterior teeth and if they brux laterally marked matching wear facets are seen affecting the upper and lower canines (if the patient has a canine guided occlusion), and wear facets may be seen on the premolars and molars if the patient has a group function occlusion. With more advanced wear a patient may 'convert' themselves from

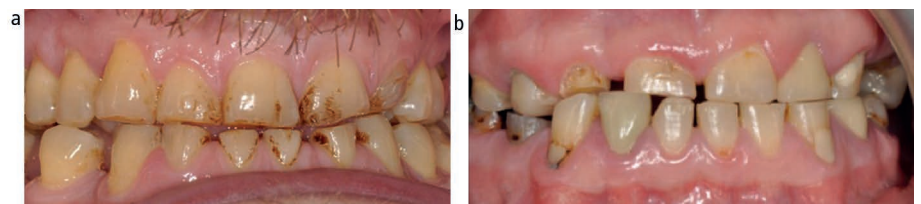


Fig. 1 Examples of attrition

a canine guided occlusion to a group function type occlusion, once wear of the canines allows contact of the posterior teeth in lateral excursion.

It is also important to realise that erosion may be superimposed or coexist with attrition/abrasion and this is often seen with patients who consume significant numbers of oranges for example. The citric acid within the oranges<sup>2</sup> can cause erosion while the fibrous structure of the fruit can cause abrasion and this is often seen on lower molars. In this scenario, once the occlusal dentine is exposed then the tooth wear may accelerate as dentine tends to be lost two to five times faster than enamel.<sup>3</sup>

## Diagnosis

The signs and symptoms typically found in a patient presenting with attrition are outlined below.<sup>4</sup>

## Symptoms

- Tooth grinding at night
- Jaw pain, fatigue and limited opening on waking
- Teeth feel loose (localised or generalised)
- Sore teeth or sore gums
- Headaches in the temporal region
- Grinding or clenching of the teeth while awake.

## Clinical signs

- Tooth wear and marked wear facets, particularly in protrusion or lateral excursion
- Tooth fractures – natural teeth or restorations
- Tooth mobility
- Pulp necrosis – as loads cause limitation of blood supply
- Traumatic ulcers
- Linear alba.

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- Masticatory muscle hypertrophy – particularly masseter and temporalis muscles
- Tongue indentations.

## Aetiology

There are generally thought to be three principal theories regarding the aetiology of attrition. In addition, there may also be modifying factors (often lifestyle factors) present, such as bone chewing.

The theories of attrition are:<sup>5</sup>

- Functional theory
- Parafunction initiated by occlusal interferences
- Central nervous system aetiology.

## Functional theory

This suggests that tooth wear occurs due to prolonged contact of the teeth and the patient having a broad envelope of function. The seminal work of Lundeen *et al.*<sup>6</sup> showed that some patients exhibit a very extensive range of movement in their usual chewing pattern, analogous to a cow chewing which leads to attrition and tooth wear.

Kim *et al.*<sup>7</sup> explored this idea further and compared a group of 15 patients with a normal 'chopping' pattern of mastication with a group of 15 subjects who had a broader 'grinding-chewing' pattern of movement. They found that the broader grinding type chewing pattern had significantly greater levels of occlusal wear compared to the 'chopping' type. It is also worth noting that this study was undertaken in a group of young patients 23–25 years old. It is therefore likely that any differences in the occlusal wear patterns seen between the 'grinding' and 'chopping' group would only increase as they aged.

## Parafunction initiated by occlusal interferences

The theory that parafunction can be initiated by occlusal interferences and therefore

managed clinically by occlusal adjustments or extensive rehabilitations has been present in the literature for many decades. Unfortunately, the evidence in the literature does not support this theory. For example, Clark *et al.*<sup>8</sup> reviewed a large number of animal and human studies and found that occlusal interferences could not cause bruxism or stop it. More recently a systematic review found no link between occlusal interferences and bruxism.<sup>9</sup>

## Central nervous system aetiology

Over the last two to three decades it has become evident that the majority of bruxism is caused by a central nervous stimulus and a great deal of work in this area has been undertaken by Professor Gilles Lavigne and his co-workers at the University of Montreal. It appears that bruxism can occur either when the patient is awake (awake bruxism) or when the patient is asleep (nocturnal bruxism).

In awake bruxism the patient is naturally aware of jaw clenching and this is a very common phenomena with a prevalence of around 20%.<sup>10</sup> The aetiology of awake bruxism is poorly understood but known risk factors are psychological stress and anxiety.

In contrast, nocturnal bruxism is tooth grinding while the patient is asleep and the patient may be aware of this, or more likely, the patient's partner or family members are aware of this problem.

The prevalence of nocturnal bruxism is reported as being 8–10%<sup>10</sup> and it has now been classified as a sleep-related movement disorder. Essentially, sleep bruxism occurs following sleep-related micro-arousals that originate in the brain stem. These micro-arousals cause the heart rate to increase following which brain activity increases. This is followed by activation of the suprahyoid muscle and then this is followed by rhythmic masticatory muscle activity resulting in bruxism.

It therefore appears that bruxism is a neurological problem and the tooth damage that we

see as dental professionals is a consequence of a neurologically initiated activity manifesting as grinding and tooth surface loss. Unfortunately, at the moment there is no effective pharmacological management for attrition but this is something that may be developed in the future.

## Modifying factors in attrition

In addition to the causes of attrition outlined above, there are a number of other factors that may be present that will accelerate the amount of tooth tissue loss.

## Ecstasy

Around 1.5% of the population use ecstasy (MDMA 3, 4-methylenedioxyamphetamine) and it is the second most popular recreational drug in 16–24 year olds. The main side effects of this drug are bruxism and a profound xerostomia that last for around 6–8 hours. Milosevic *et al.*<sup>11</sup> reported that within a group of regular ecstasy users, 60% showed wear into dentine versus 11% of non-users and 89% of the ecstasy users complained of tooth grinding and over 90% of users reported having a dry mouth and consuming three cans of carbonated drinks 'per trip'. This is a good example of a combined attrition and erosion aetiology.

## Habitual chewing on hard food stuffs

Habitual chewing on hard foods or unusual food stuffs such as bone chewing. Bone chewing to release the marrow is particularly common in ethnic Chinese<sup>12</sup> and this may also exacerbate tooth surface loss.

## Selective 5-hydroxytryptamine reuptake inhibitors (SSRIs)

It is estimated that around 7% of the UK population are taking antidepressants<sup>13</sup> and the commonest type of antidepressants used to manage anxiety and depression are the SSRI group which increase levels of the neurotransmitter 5-hydroxytryptamine in the brain. Recent reports<sup>14,15</sup> suggest that SSRIs may cause bruxism, particularly when patients first start taking these drugs and that it may be worthwhile talking to the patient's medical practitioner about changing the SSRI for an alternative drug.

## Lack of posterior support

Many clinicians consider that a lack of posterior support can eventually lead to more tooth wear on the remaining anterior teeth. Initially, this seems logical as patients without any posterior

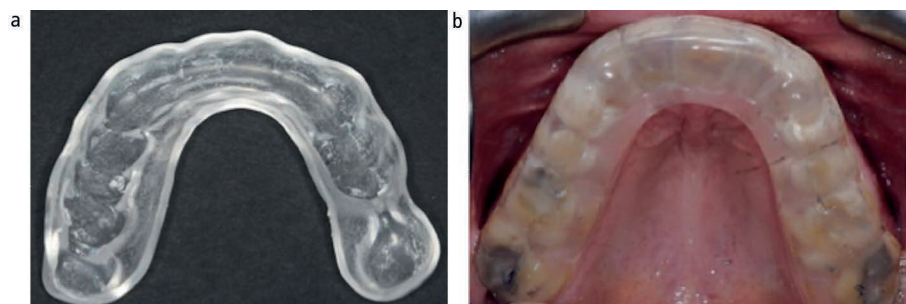


Fig. 2 Hybrid hard and soft splint

teeth will tend to do all of their chewing with their anterior teeth. However, things are not as straightforward as this.

Kozawa *et al.*<sup>16</sup> tested a small group of patients (N = 5) receiving posterior bridgework and provided test restorations where the molars and premolars were omitted from the bridges one tooth at a time. They found that the maximum bite force on the anterior teeth actually decreased rather than increasing as more and more posterior teeth were removed. This is most likely to be a reflection of the mandible acting as a second class lever when it is being used to crush food on the molars – here the load is greatest nearer to the condyle in the molar region and the loads will be much less on the anterior teeth.

Smith and Robb<sup>17</sup> reviewed tooth wear in a large sample of 1007 patients and found no relationship between the number of missing posterior teeth and anterior tooth wear while Wazani *et al.*<sup>18</sup> examined 290 patients referred to a teaching hospital and reported a very strong relationship between lack of posterior support and wear of anterior teeth (which was statistically significant at  $P = 0.001$ ).

Unfortunately this is still rather a confused area within the literature and is worthy of further investigation.

## Clinical management

The clinical management of attrition is potentially problematic as the restorative treatment will not cure the attritional tendencies of the patient, as the drive for this is neurological, as discussed earlier. It is therefore imperative that the patient understands the aetiology of this condition and is willing to commit to wearing a protective splint long term and potentially for a lifetime. The more complex and extensive the treatment is, the more important it is that the patient commits to this long-term splint wearing.

Treatment for bruxism can also be divided into an initial or diagnostic phase and a 'definitive' treatment phase, although it can be argued that the clinical management of a bruxism patient is never definitive as the risk for restoration failure is ever present.

It is therefore often very useful to have an initial 'diagnostic' phase of treatment where the clinician can convince him/herself that the patient is compliant with splint wearing. The choice of splints available for bruxism patients are either hard splints, soft splints and, more recently, hybrid splints with a soft inner lining and a hard outer shell (Fig. 2). Splints can be

used in either the upper or lower arch but there is little guidance in the literature as to which is best. Some clinicians believe it is potentially better to use a splint in the upper arch as the hard palate is available to distribute any occlusal loads more widely, but hard evidence for this recommendation is lacking.

Hard (usually acrylic) splints (Fig. 3) can also be used to allow any spasm in the muscles of mastication to resolve, which will allow recording of the retruded contact position (RCP) more easily. In addition to this, a hard splint can also be used to reversibly assess any increase of the occlusal vertical dimension if this is thought necessary, although many clinicians will now only use a splint in this way if increases in OVD are large and in excess of 4–5 mm.

During this initial diagnostic phase, it is also important to explore any other potential contribution to the tooth wear presenting clinically, such as erosion. This may well involve the use of a diet history taken over at least three days<sup>19</sup> together with the use of topical fluoride<sup>20</sup> and modern desensitising toothpastes.<sup>21</sup> If toothbrush abrasion is thought to be a contributory factor then advice about brushing in relation to consumption of any acidic food or drink at least an hour apart should be given.<sup>21</sup>

## Restoration

Once the clinician is comfortable that the patient is wearing his/her splint regularly then consideration can be given to restoration of the worn teeth. Usually, the main driver for this is that the patient is concerned about the appearance of their short upper anterior teeth and the aesthetic impact that this is having.

This wear may be isolated to the anterior teeth, which may affect either the upper or lower anterior teeth in isolation, or the upper or lower anterior teeth in combination. Alternatively there may be more generalised wear affecting both the anterior and the posterior teeth and this wear may be localised to either the maxillary or the mandibular arch, or in the most severe cases both the arches.

A popular technique for managing localised anterior tooth wear is the Dahl technique first described by Dahl and his co-workers in 1975.<sup>22</sup> The main problem with tooth wear, including attrition, is that it usually takes place slowly<sup>23</sup> with a typical loss estimated to be less than 15 microns per year. As a result of this, in most patients the teeth move into the space created by the tooth wear in a process known



**Fig. 3** Hard splint

as compensatory overeruption.<sup>24</sup> As a consequence the main issue in many tooth wear cases is a lack of space anteriorly in which to place restorations such as crowns.

The Dahl technique was designed to overcome this problem by placing what was essentially a metal anterior bite raising platform which the patient wore for around 6–12 months.<sup>22,25</sup> This bite platform causes disclusion of the posterior teeth and over a period of 6–12 months the posterior teeth erupt back into contact at an increased OVD dictated by the Dahl appliance. In addition to the posterior tooth eruption, there is also an element of intrusion of the lower anterior teeth caused by the appliance.<sup>26</sup> Since the Dahl technique was first described it has gone through many iterations clinically<sup>27</sup> and currently the most popular way of undertaking this approach is to restore the worn palatal surfaces and replace the missing tooth structure with direct composite resin to produce a Dahl effect without the need to use a metal Dahl appliance first.<sup>28,29</sup> The beauty of this technique is that once the OVD is increased by the addition of palatal composite, the worn incisal edges can be restored to their original length and many clinicians, including the authors, would advocate placing an extensive labial bevel within enamel to maximise retention and produce what is in essence a ferrule effect.

More recently, many clinicians have extended this technique with composite resin to manage both upper and lower anterior tooth wear together with the so called 'double Dah' technique (Fig. 4) where both the upper and lower anterior teeth are restored simultaneously.<sup>30</sup>

A number of studies have investigated the longevity of the direct composite resin Dahl approach and these have shown that these restorations are successful in the medium term.<sup>31–35</sup> The study of Gulamali *et al.*<sup>34</sup> has provided the longest follow up period at ten years and found that there was a 50% probability that composite restorations would survive for seven years, but 90% of restorations showed either minor failure (chipping/staining) or major failure (cracking) by ten years. However,





Fig. 4 Double Dahl technique

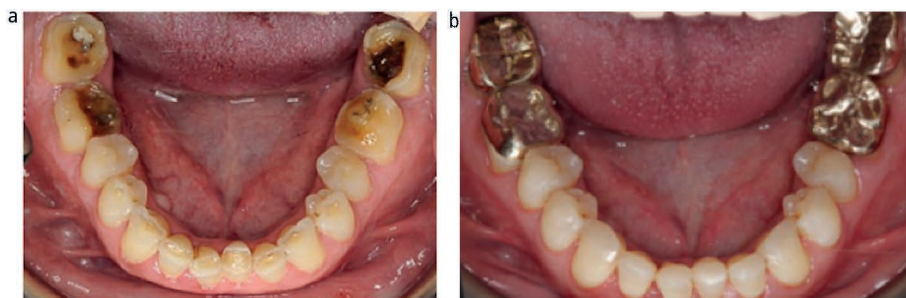


Fig. 5 Gold onlays used to manage generalised attritional wear

the patients followed in this sample would have had a mixture of both erosive and attritional tooth wear, and it is therefore likely that the patients treated with the Dahl technique with attrition would have showed a higher rate of failure. The potential adverse consequences of repeated re-restoration of worn teeth is loss of pulpal vitality and reduced bond effectiveness. However, a major advantage of this technique of this approach is that in most instances the composite restorations can be repaired simply and quickly.

Localised upper anterior tooth wear may also be managed using adhesive/dentine bonded crowns utilising the Dahl approach<sup>36,37</sup> but fracture remains a risk, so again a protective occlusal splint is mandatory.

Generalised wear can also be managed using direct composite restorations and clinical techniques to undertake this have been described by Attin *et al.*,<sup>38</sup> Smales and Berekally,<sup>39</sup> and Milosevic & Burnside.<sup>40</sup> Attin *et al.*<sup>38</sup> describe a technique using clear silicone putty matrices for restoring the posterior teeth developed from an initial diagnostic wax up and following this the anterior teeth are restored. Attin *et al.*<sup>38</sup> have also followed a small group of six patients treated in this way and the reconstructions have survived well to 5.5 years. However, it is important to note that all the patients in this very small prospective case series were erosive tooth wear cases. There are therefore obvious concerns about the transferability of these findings to a cohort of patients with bruxism.

An alternative and conservative approach to managing full arch tooth wear is to use

adhesive gold onlays on the molar teeth and use direct or indirect composite to restore the remaining teeth, increasing the OVD as necessary (Fig. 5). Adhesive gold onlays are rather more conservative than traditional gold crowns and Chana *et al.*<sup>39</sup> reported an 89% survival probability at five years when cemented with Panavia cement (Kuraray Dental, 33 Maiden Lane, New York, USA).

Finally, the more traditional way of dealing with a full arch or mouth rehabilitation is to use conventional crowns. These are usually placed using a mutually protected occlusal approach with canine guidance and anterior guidance protecting the posterior teeth in protrusive jaw movements.<sup>40</sup> This traditional approach is both very destructive and very expensive. There is a relatively high possibility of pulpal death in around 10–15% of teeth<sup>41</sup> and the longevity data for conventional crowns suggests a mean life span of around ten years.<sup>42,43</sup> However, it is important to appreciate that the longevity data is based on pooled data for both bruxists and non-bruxists. As the prevalence of bruxism is around 10% then the longevity data for conventional crowns may well be less than ten years in patients with a history of bruxism.

## Conclusions

Patients with attrition, particularly when caused by bruxism, are arguably the most challenging of the 'tooth wear' patients to manage. With this group of patients it would seem a sensible approach to exhaust the possibility of using more conservative adhesive approaches

first, as this minimal preparation approach preserves both tooth structure<sup>44</sup> and pulpal vitality.<sup>43</sup> An occlusal splint that the patient is comfortable wearing is almost always required and more conventional techniques can be kept in reserve for when they are absolutely necessary.

1. Paesani D A. Toothwear. In Paesani D A (ed) *Bruxism – Theory and Practice*. pp. 123–147. Quintessence Publishing Co. Ltd., New Malden, Surrey UK, 2010.
2. Rees J.S, Burford K, Loyn T. The erosive potential of alcoholic lemonades. *Eur Journal Pros Rest Dent* 1999; **6**: 161–164.
3. Hooper S, West N X, Pickles M J, Joiner A, Newcombe R G, Addy M. Investigation of erosion and abrasion on enamel and dentine: a model *in situ* using toothpastes of different abrasivity. *J Clin Periodontol* 2003; **30**: 802–808.
4. Paesani D A. Diagnosis of bruxism. In Paesani D A (ed) *Bruxism – Theory and Practice*. pp. 21–40. Quintessence Publishing Co. Ltd., New Malden, Surrey UK, 2010.
5. Spear F. Treating the worn dentition. The essential Frank Spear DVD Seminar Series, 2012. Spear Institute, Scottsdale, Arizona USA.
6. Lundeen H.C, Shryock E.F, Gibbs C H. An evaluation of mandibular border movements: their character and significance. *J Prosthet Dent* 1978; **40**: 442–452.
7. Kim S K, Kim K N, Chang I T, Heo S J. A study of the effects of chewing patterns on occlusal wear. *J Oral Rehabil* 2001; **28**: 1048–1055.
8. Clark G T, Tsuiyama Y, Baba K, Watanabe T. Sixty-eight years of experimental occlusal interference studies: what have we learned? *J Prosthet Dent* 1999–**82**: 704–713.
9. De Boever J A, Carlsson G E, Klienbergh I J. Need for occlusal therapy and prosthodontic treatment in the management of temporomandibular disorders. Part I. Occlusal interferences and occlusal adjustment. *J Oral Rehabil* 2000; **27**: 367–379.
10. Lavigne G.J, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: an overview for clinicians. *J Oral Rehabil* 2008; **35**: 476–494.
11. Milosevic A, Agrawal N, Redfern P, Mair L M. The occurrence of toothwear in users of Ecstasy (3, 4-methylenedioxyamphetamine). *Comm Dent Oral Epidemiol* 1999; **27**: 283–287.
12. Milosevic A, Lo M S. Tooth wear in three ethnic groups in Sabah (North Borneo). *Int Dent J* 1996–**46**: 572–578.
13. Wilson C. High antidepressant use could lead to UK public health disaster. 2016. Available at <https://www.newscientist.com/article/2087949-high-antidepressant-use-could-lead-to-uk-public-health-disaster/> (accessed July 2016).
14. Milanloglu A Paroxetine-induced severe sleep bruxism successfully treated with buspirone. *Clinics* 2012; **67**: 191–192.
15. Lineberry J. Link Between Medications and Bruxism? 2017. Available at <https://www.speareducation.com/spear-review/2013/03/link-between-medications-and-bruxism> (accessed July 2017).
16. Kozawa T, Igarashi Y, Yamashita S. Posterior occlusal support and bite force influence on the mandibular position. *Eur J Prosthodont Rest Dent* 2003; **11**: 33–40.
17. Smith B.G, Robb N D. The prevalence of toothwear in 1007 dental patients. *J Oral Rehabil* 1996; **23**: 232–239.
18. Wazani B E, Dodd M N, Milosevic A. The signs and symptoms of tooth wear in a referred group of patients. *Brit Dent J* 2012; **213**: E10.

19. Kidd E A, Smith B G N. Toothwear histories: a sensitive issue. *Dent Update* 1993; **20**: 174–178.
20. Mohammed A, Durasa K. What is the role of topical fluoride application in preventing dental erosion? *Evid Based Dent* 2013; **14**: 59–62.
21. Chander S, Rees J S. Strategies for the prevention of erosive tooth surface loss. *Dent Update* 2010; **37**: 12–14, 16–18.
22. Dahl B L, Krogstad O, Karlsen K. An alternative treatment in cases with advanced localized attrition. *J Oral Rehabil* 1975; **2**: 209–214.
23. Rodriguez J M, Austin R S, Bartlett D W. *In vivo* measurements of tooth wear over 12 months. *Caries Res* 2012; **46**: 9–15.
24. Berry D C, Poole D F. Attrition: possible mechanisms of compensation. *J Oral Rehabil* 1976; **3**: 201–206.
25. Gough M B, Setchell D J. A retrospective study of 50 treatments using an appliance to produce localised occlusal space by relative axial tooth movement. *Br Dent J* 1999; **87**: 134–139.
26. Chadwick R G. *Dental Erosion*. Quintessence Publishing Co. Ltd., London, 2006.
27. Briggs P F, Bishop K, Djemal S. The clinical evolution of the 'Dahl principle'. *Br Dent J* 1997; **183**: 171–176.
28. Mizrahi B. A technique for simple and aesthetic treatment of anterior tooth wear. *Dent Update* 2004; **31**: 109–114.
29. Saha S, Summerwill A J. Reviewing the concept of Dahl. *Dent Update* 2004; **31**: 442–447.
30. Poyser N J, Porter R W J, Briggs P F, Chana H S, Kelleher M G. The Dahl concept: Past, present and future. *Br Dent J* 2005; **198**: 669–676.
31. Gow A M, Haemmings K W. The treatment of localized anterior tooth wear at an increased occlusal vertical dimension. Results after two years. *Eur J Prosthodont Restor Dent* 2002; **10**: 101–105.
32. Haemmings K.W, Darbar U.R, Vaughan S. Tooth wear treated with direct composite restorations at an increased vertical dimension: results at 30 months. *J Prosthet Dent* 2000; **83**: 287–293.
33. Redman C.D, Haemmings K.W, Good J A. The survival and clinical performance of resin-based composite restorations used to treat localised anterior tooth wear. *Br Dent J* 2003; **194**: 566–572.
34. Gulamali A B, Haemmings K W, Tredwin C J, Petrie A. Survival analysis of composite Dahl restorations provided to manage localised anterior tooth wear (ten year follow-up). *Br Dent J* 2011; **211**: E9.
35. Al Jawad A, Rees J S. Retrospective study of the survival and patient satisfaction with composite Dahl restorations in the management of localised anterior tooth wear. *Eur J Pros Rest Dent* 2016; **24**: 222–229.
36. Satterthwaite J D. Indirect restorations on teeth with reduced crown height. *Dent Update* 2006; **33**: 210–216.
37. Burke F J T. Four year performance of dentine-bonded all-ceramic crowns. *Br Dent J* 2007; **202**: 269–273.
38. Attin T, Filli T, Imfeld C, Schmidlin P R. Composite vertical bite reconstructions in eroded dentitions after 5-5 years: a case series. *J Oral Rehabil* 2012; **39**: 73–79.
39. Smales R J, Berekally T L. Long-term survival of direct and indirect restorations placed for the treatment of advanced tooth wear. *Eur J Prosthodont Rest Dent* 2007; **15**: 2–5.
40. Milosevic A, Burnside G. The survival of direct composite restorations in the management of severe tooth wear including attrition and erosion: A prospective 8-year study. *J Dent* 2016; **44**: 13–19.
41. Chana H, Kelleher M, Briggs P, Hooper R. Clinical evaluation of resin-bonded gold alloy veneers. *J Pros Dent* 2000; **83**: 294–300.
42. Chu F C S, Siu A S C, Newsome P R H, Chow T W, Smales R J. Restorative management of the worn dentition. 4: Generalized tooth wear. *Dent Update* 2002; **29**: 318–324.
43. Cheung G S P, Lai S C N, Ng R P Y. Fate of vital pulps beneath a ceramic metal crown [CMC] or a bridge retainer. *Int End J* 2005; **38**: 521–530.
44. Edelhoff D, Sorensen J A. Tooth structure removal associated with various preparation designs for anterior teeth. *J Prosthet Dent* 2001; **87**: 503–509.